THE HISTORY OF A PAIR OF EYES

BY

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DEDICATED

TO

THE GENTLEMEN OF

THE AMERICAN MEDICAL ASSOCIATION

WHO

HAVE EVER STOOD READY TO ASSUAGE HUMAN SUFFERING

REGARDLESS

OF PERSONAL RISK AND SACRIFICE

NOT FOR ACCEPTANCE BUT FOR CONSIDERATION

PREFACE.

In publishing the statistics of my eyes, I wish to say that I am not actuated by any desire to reflect upon the physicians in whose hands my case has been. Far from it, the results were due, not to their lack of skill, but to the condition of my eyes; and it is to demonstrate this truth, to intensify this fact, that, in dealing with these spasms, we are confronted, not with static but with dynamic quantities, I insert the formulæ.

I mention the severe effect of atropine because it is in harmony with the views held by me in regard to the eye. I explain it as due to the fact that I had an elastic cramp of the ocular muscles. The atropine decomposed it and set it in vibration.

In calling attention to the fact that the tests were made with lens, ophthalmoscope, and retinoscope, it is not to insinuate that the physicians blundered, but to emphasize a much deeper truth — that the instruments themselves are unreliable.

I think the consideration of the eye from the position assumed by me will harmonize the diverse results as

found by Dr. Stevens and his opponents. To illustrate, two men may have the same apparent weakness of the extrinsic muscles. In one, it produces severe constitutional effects; the other bears it with comfort and ease. As long as we consider alone the strain upon the apparent weak muscles we are confronted with a phenomenon we cannot explain, but it becomes readily explicable when we consider the cramps of the antithetical muscles, according to their nature and consistency, whether they are rigid or elastic, whether they remain firm or readily go into vibration.

INTRODUCTION.

The body of the following article is a curtailment from a slightly longer article written by me in the winter of 1894, which at some future date it may be deemed advisable to publish in full. It is simply what it professes to be, "The History of a Pair of Eyes," with such deductions drawn as seemed warranted. An effort has been made by artificial experiments to determine whether it is the nervous condition affecting the eye or whether it is the eye producing the nervous condition. These experiments will be found in the Appendix, and will be allowed to speak for themselves.

Where it has been found necessary to criticise work done, it is a criticism of principles and not of persons, the idea being to throw light on the subject and not to cast reflections.

The errors to which attention has been called are the insufficient correction of the astigmatism, its correction at a faulty angle, the injudicious use of heavy, unaccepted sphericals, and the injudicious use of atropine. The new ideas contained are, that what is commonly regarded as

lack of balance of the ocular muscles is generally a cramp of the strong muscle; that so-called paresis of the external is in reality a cramp of the internal, and the recognition, not only of a cramp of the muscles, but an elastic cramp giving rise to spasm.[1]

In dealing with that which is new judgment is best displayed by conservatism rather than radicalism; still, I think myself assured in assuming that those conditions of muscular asthenopia which are combined with an elastic cramp of the ocular muscles will throw material light in some cases of such diseases as chorea, epilepsy, hysteria, and heart-failure, and that the sudden breaking of this cramp may be attended by fatal results in which the real primary disturbance may be masked by a distant reflex.

I have stated that they are liable to decomposition through the assimilation of certain foreign substances. Is it advisable to use those particular drugs which have this effect? Those cases in which the drug is used for its constitutional effect belong to the science of therapeutics, with which I have nothing to do, though it would be well

[1] For the purpose of clearness of definition, I think it necessary to increase the nomenclature of ophthalmology by the addition of the term cramp and the limitation of meaning of the term spasm. Heretofore the term spasm, as applied to the accommodation, has been used to designate two separate conditions – tonic contraction, a static condition; and spasmodic oscillation, a dynamic one. I have used the terms cramp and spasm respectively to designate the distinction.

for the practitioner to bear in mind their incidental effect or he may produce serious eye-complications. To limit the question, is it advisable to use those particular drugs having this effect for the sole object of decomposing these cramps? Even in this the field is too new to give a definite answer. All I can do is to present the conditions. From my position I regard stability, firmness, and rigidity as of prime importance, and it remains to be determined whether success would attend such efforts or whether we would merely render the cramp elastic and intensify instead of ameliorate the conditions.

In the Appendix certain experiments are given whose object it is to demonstrate, by artificially augmenting strains, that certain severe reflexes can be produced from the eye. If, now, by purely mechanical means, these reflexes can be produced, they are coming from the eye, no matter how the eye got into that condition, whether it be the cramps were never firmly hardened, whether it be they have been weakened by toxic drugs or a spell of sickness, and if it be true that such severe reflexes can be produced in such a short space of time by slightly augmented strains, it becomes readily explicable how the slow accretion of minor strains, from minute to minute during the day may produce almost any degree of neurasthenic depletion.

I confess that the feelings I experience in penning this introduction are less sanguine than those experienced in

writing the body of the article last winter. It was only on maturer views that I recognized the seriousness due to the elastic condition of the muscles as well as to position. The treatment of this condition is not within the sphere of the optician. His mission is finished when he has corrected the refractive error and balanced the muscles as near the point of equilibrium as possible. The future treatment of the condition, the decomposition of the cramp, if deemed advisable, is the function of the judicious physician, though there remains the uncertainty of whether or not it can be successfully attained.

THE HISTORY OF A PAIR OF EYES.

I.

THE UNCORRECTED ERROR.

THE ACQUIRED LENTICULAR ASTIGMATISM AND ITS RELATION TO EYE-STRAIN AND NEURASTHENIA.

It is not the purpose of this article to deal with astigmatism in general, nor with the general problem of lenticular astigmatism, but, as its title indicates, its attention will be confined exclusively to that form of lenticular astigmatism which is only apparent and not real, and which does not generally yield to the use of atropine as ordinarily administered.

The principle, briefly stated, is this: that, where a person possesses a corneal astigmatism (and the majority of people do), which astigmatism may readily be determined by the ophthalmometer, they will make an endeavor to neutralize this by forming an artificial lenticular astigmatism by cramping the ciliary muscles. In the majority of cases this acquired astigmatism becomes so

thoroughly latent that the use of a strong solution of atropine for a few days does not serve to separate the astigmatic meridians.

The cylindrical correction made is generally an under correction; eye-strain may continue and neurasthenia may persist.

The points to be brought out in the following article, so far as the eye is concerned, are —

1st. Atropine as ordinarily administered does not entirely separate the astigmatic meridians as a rule.

2d. The ophthalmometer correctly reads the cornea.

3d. Except when modified by a rare lenticular astigmatism, it gives the true angle and amount and the eye can be brought up to its readings.

4th. In case of a strong spasm, the last meridian to yield is often the astigmatic meridian, in consequence of which a wrong angle is often given.

5th. By a long-continued eye-strain or under various nervous shocks the astigmatic meridian may voluntarily separate, the tendency becoming greater as the accommodation weakens. In cases a local chorea, or spasmodic oscillation of the ciliary muscle may ensue, caused by the attempt, on the one hand, to obtain distinct vision, and, on the other, by the natural tendency of the muscles to fall in their proper positions.

6th. This oscillation in its fainter forms is imperceptible to the ordinary tests; in its most advanced forms it shows

itself outwardly in the jumping of type and the blurring of vision. It becomes a direct irritation to the branch of the third cerebral nerve, and may reflex with grave consequences to various portions of the body.

7th. $\frac{20}{20}$ is not the test of vision of the normal eye at its best.

8th. Eye-strain, sufficient to produce reflex nerve action, may exist without any marked sensation in the eye itself.

9th. Latent errors of refraction, like a voluntary effort of accommodation, may act upon the convergence of the eyes, producing what is known as lack of balance of the ocular muscles. This strain, entailed on the muscles by the effort, on the one hand, to assume the normal relation with accommodation, and, on the other, to secure distinct binocular vision, may produce a spasm of the ocular muscles, which may reflect with terrific force on the nervous system.[1]

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[1] Perhaps no place is more fitting than the present for giving my full views in regard to these ocular cramps. Regarded as primarily due to latent errors of refraction, they become more or less solidified. I regard the primary cramps of the external and internal, in myopia and hyperopia respectively, as dependent upon consensual relation, which is in harmony with the well-known biological law that that which is habitually associated in action in the history of an organism becomes functionally united in relation. I regard it as possible to form a secondary cramp in the antithetical muscle from the habitual act of securing binocular vision, giving us such exceptional cases as double cramps. They are subject to strains of various natures. They are all subject to the strain in the attempt to secure binocular vision. The cramp of the interni are subject to the strain of the attempt at divergence of the

For convenience of reference I here prefix the various prescriptions as found for my own eyes. It will be well to state here that the meridians in the right eye cross at 60° and 90°, with a hyperopia of plus 4 D^s and an uncorrectable astigmatic separation of plus 0.5 D^c. It has played little part in vision and none in reflex nerve action. It may therefore be eliminated from the article.

O. S. = — .25 D^s. October 2, 1882. Physician A.	Without atropine. Test by lens and ophthalmoscope.
O. S. plus $\frac{1}{24}$ D^s — $\frac{1}{42}$ D^c, axis, 180°. February 15, 1884. Oculist B.	Under atropine, strength solution unknown, injected three times day of examination, during which there were marked constitutional effects. Tests by lens and ophthalmoscope. (For reading.)

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eyes in conforming with the relaxation of the accommodation as it weakens.

They are liable to decomposition from any disease that is capable of seizing upon them. They are liable to decomposition from the assimilation of certain substances.

Granting to some of them a degree of elasticity and we have all the data necessary to furnish us with various spasmodic motions of the body.

They may be so delicately poised as to be in a state of almost continuous spasm.

According to their degree of elasticity, they are liable to go into vibration under any of the above-mentioned strains, or under any draught upon the nerve energy, either psychological or physical.

This and all other notes are added since the writing of the body of the article.

O. S. Prisms, 2° base out. O. D. August 19, 1885. Oculist C.	Without atropine. Tests by lens and ophthalmoscope. (To be worn over Reuling's glasses for reading.) S. M. Burnett, M.D., August 19.
Oculist D.	Without atropine. Test by lens and ophthalmoscope. Reuling's glasses sanctioned for reading. Prisms discarded and faradic current electricity used daily.
O. S. = plus 1.5 D^s, plus 0.5 D^c, axis 60°. January 25, 1887. Oculist E.[1]	Under atropine, two grains to ounce water, once, at bedtime, five days previous examination. Test by lens and ophthalmoscope. (To be worn constantly.)
O. S. = plus 0.25 D^c, axis unknown. 1888. Physician A.	Without atropine. Test by lens and ophthalmoscope. (To be worn constantly.)
O. S. = plus 0.5 D^c, axis, 60°. December 9, 1889. Oculist E.[1]	Circumstances examination forgotten. (For distance.) Glass of 1887 to be worn for reading.
O. S. O. D. Prisms, 3° base out. January 15, 1890. Oculist E.	(To be worn over specs.)
O. S. plus 1.5 D^s, plus 0.5 D^c, axis 105°. May 24, 1890. Oculist F.	Under atropine, quantity unknown. Test by lens, ophthalmoscope and retinoscope. (To be worn constantly.)

[1] O. D. + 4. D^s + 1 D^c, axis 90°.
O. D. + 2. D^s + 1 D^c, axis 90°.

Amount of cutting internal recti muscles, 12°. Superior rectus right eye, and inferior rectus, left eye, 5°. Summer, 1890. Oculist F.	Operated on four muscles for esophoria and hyperophoria.
O. S. plus, 2 D^s plus 0.75 D^c, axis 100°. September 6, 1890. Oculist F.	Under atropine, two grains to ounce water, three times per day for two days previous to examination, and an injection of a three per cent. solution just prior to the examination followed by grave constitutional symptoms. Test by lens, ophthalmoscope, and retinoscope.
O. S. plus 1.62 D^s plus 1.50 D^c, axis 100°. November 16, 1893. By myself.	Under atropine, two grains to the ounce, for six days, twice per day, and wearing plus 2 D^s plus 0.75 D^c, axis 100°.
O. S. plus 1.62 D^s plus 1.75 D^c, axis 100. November 30, 1893.	Atropine continued through December 1st, same strength solution, once per day, and the prescription of November 16th placed upon the eye November 29, 1893.
Plus 1.75 D^c and plus 2 D^c, axis 100°, giving same result on astigmatic dial. December 1, 1893. By myself.	[1]
Plus 2 D^c, axis 97°. Oculist G.	Corneal astigmatism as read on the ophthalmometer.

[1] This illustrates the method formulated by me for proving and testing the correctness of ophthalmometric readings, to continue the use of atropine and crowd the eyes with both spherical and cylinder. A better illustration is given later on.

The symptoms of eye-strain had become pronounced early in life. In 1882 the left eye had cramped itself in to accommodate myopia. At this day it is impossible to remember the symptoms with accuracy further than to say at times there was a pain in the eyeball and an occasional blurring of vision, a frequent dreamy, hazy feeling, fulness of the head, and some discomfort of the eyes after protracted strain. In 1884 these feelings had become intensified, together, occasionally, after long reading or great physical fatigue, that has been called the "premonition of approaching insanity."

Among other symptoms that may be noted was a feeling of weakness, going so far at times as to cause a trembling of the knees when at my studies, a feeling of nervousness and a frequency of urination. The passionate nature was unduly aroused and sexual weakness displayed itself early in life. I lay stress on these undue sexual symptoms as I wish to refer to them again.

The glass of 1884 produced some relief for awhile. I think the error was in not making a cylindrical correction for distant vision, but in 1885 the symptoms again became marked, so much so that during the last quarter at school I found it necessary to give up Greek on account of the type.

The prisms of 1885 were without avail. The electric battery caused a temporary relief of the symptoms, a material clearing of the head, immediate in its effect, and an

increased power of the eyes. By November, 1885, they had picked up so much that I started back at school for a post-graduate course, and remained until March. My eyes had again commenced to trouble me, and it was at this time I found it necessary to almost wholly give up reading after dark.

Notwithstanding that my eyes were getting worse, in November, 1886, I matriculated from the Columbian Medical College, Washington, D. C. I had been studying hard and type had commenced to blur quite badly. A few days later I suffered a nervous shock from visiting the dissecting-room. From this moment the unpleasant sensations increased with frightful rapidity. The type commenced to blur worse, a dreamy, hazy, cloud-like feeling settled down, nervousness was more intense and the feeling of approaching insanity never absent. The sense of effort which had been increasing with every intellectual attempt for the last four years, kept on increasing. My studies were only acquired by intense effort and persistent repetition.

The climax came December 17, 1886, while writing at my desk, with the type blurring before my eyes, every now and then brushing my hand across and holding it over them for a few moments, thus attaining temporary relief, I felt as if a rush of blood on the left side in the neighborhood of Meckel's ganglion. In a few minutes a tight band-like feeling had encircled the head, running

around the brow, back across the temples and around the lower back portion of the head, just above the neck. A peculiar gnawing sensation, as if of a splinter twisting itself in the brain, commenced on the left side, in the neighborhood of where I felt the spurt of blood, as it seemed. The whole rear portion of the head seemed like a burning coal of fire. A dull pain manifested itself in the left ear. Vertigo commenced, which was not to leave for nearly a year. Then commenced all the horrible subjective sensations of a well-developed cerebral congestion; symptoms for which the terms ordinarily used in medical works bear no adequate description. It is my intention at some time to amplify a description of these sensations. It is sufficient here to adopt the current terms and describe it as the "haze of melancholia," "nervous hesitation" and "the premonition of approaching insanity," and a feeling of approaching delirium. This last symptom lasted for perhaps two or three years. The others endured for seven.[1]

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[1] The question naturally arises, what occurred at this time. I do not propose to answer that question directly, but I call attention to the fact that among other symptoms was a peculiar gnawing sensation on the left side, in the neighborhood of Meckel's ganglion, which endured for a long time. It was undoubtedly an integral part of my sickness, either as a symptom or a cause. Now, as shown in the Appendix, I have been able to artificially produce this by mechanical manipulations on the eye, and can still do so at will. If it can be produced as a neurotic reflex from the eye now, and all the conditions were present years ago which might have produced it, I see no reason for assuming that it was other than a reflex from the eye then.

It was in this condition that I went into the hands of Dr. S. O. Richey. He has since said that when I entered his office, I looked like a maniac. I have no doubt the description is just, for my feelings approximated very nearly that condition. Objects were shaking and vibrating. It was impossible to gain a clear or distinct view of anything. Not only was it almost impossible to look the physician straight in the face, which he regarded as a diagnosing symptom, but it was torture to fasten my gaze on any object. Photophobia was present in its most severe form, and the test with the opthalmoscope was almost unendurable. I remember the test turning the physician dizzy on account of the rapid change of my own accommodation. I regret being unable to give the notes of this examination. I think my vision was about $\frac{20}{200}$. Under atropine and with the glass vision was brought up to $\frac{20}{20}$, and objects had become fairly steady. After the atropine passed out, objects again commenced to jump, and it was, perhaps, two years before I could distinguish anything with clearness of outline. After the disappearance of the effect of atropine, all the symptoms returned in their most severe form. Insomnia and the flashing of light before the eyes in a dark room lasted for years. For five years I could not average more than fifteen minutes' reading per day, and for the last two perhaps not more than an hour per day.

Then followed various courses of treatment by different

physicians. It is, perhaps, fitting to say that in addition to the oculists before mentioned, I was in the hands of four nerve experts and six general practitioners—the range of diagnoses extending through melancholia, hysteria, neurasthenia, dyspepsia, cerebral hyperæmia, cerebral anæmia, etc.

It is unnecessary to speak further of the treatment than to say that the mental symptoms were only palliated to the slightest degree, and I regard that as due to the effect of time and the almost entire disuse of the eyes. For seven years I hung suspended on the abyss of insanity, each mental effort seeming to still further strain the delicate cords which bound together the coherency of my mental actions.

The physical symptoms became better. The stomach, which had been very weak, picked up. The burning pain left the back of the head after a couple of years, to be succeeded by a heavy, dull pain, which also finally disappeared. The band-like stricture left the temples, the gnawing sensation in the neighborhood of Meckel's ganglion first gave place to a feeling as if some one had touched the key of an electric battery and then disappeared. The dull pain in the left ear and the feeling in the throat, which I cannot otherwise describe than as I have described it to the physicians at the time, as a metallic taste, both disappeared, though the feeling as of an electric tap, and the dull pain in the left ear, returned faintly on each attempt to use the eyes.

In the last few days of October, 1893, I started in to study the fitting of eyes for glasses. My left eye was measured on the ophthalmometer and showed something over 2 D^c, axis 100° scant. Convex cylindricals were applied and it was found the highest the eye would accept was +.75 D^c, axis 100°. Considering the heavy use of atropine which I had undergone, under the various oculists, and that I had used it myself dozens of times in the vain endeavor to get my eyes to accept my glasses, the matter was dropped on the theory of lenticular astigmatism neutralizing the cornea. (See "Errors of Refraction," Francis Valk, edition 1893, pp. 246, 247.) A few days after, in testing my eyes with various lenses, it accepted minus 1.50 D^c, axis 10°. At the same time a feeling of rest came over the eye. Had this been due to the anterior meridian forming an artificial astigmatism, it should have been accompanied by no such feeling. However that be, it made me very sceptical as to the nature of that lenticular astigmatism. The explanation which I have since had reason to give is that while the torpidity of the posterior meridian had not passed out sufficiently for it to relax, yet it had sufficient vitality to count in accommodation. I wish to draw particular attention to this phenomenon, for on this and analogous results I propose to base some conclusions in regard to accommodative myopia and strong tonic spasms.

On November 9, 1893, I started using atropine, twice

per day, two grains to the ounce of water, and wearing O. S. equals plus 2 D^s, plus .75 D^c, axis 100°. At the end of about a week the eye stood plus 1.62 D^s, plus 1.50 D^c, axis 100° equals $\frac{17}{10}$, although it had never before given above $\frac{21}{20}$. Some delay was experienced in getting the glass, during which several interesting facts were demonstrated. Atropine of same strength solution was continued once per day through to December 1st, the above glass being placed on the eye November 29th. On the morning of November 30th, the eye accepted a cylinder of plus 1.75 D^c, axis 100°, and on December 1st, plus 2 D^c and plus 1.75 D^c, axis 100°, gave the same result on the astigmatic dial.

While considering the advisability of increasing the cylinder, the reaction came.

On the night of December 1, 1893, I retired about eleven o'clock, feeling unusually well. I had been asleep a short time when I woke up with a gasp. The heart (which, I should have stated before, during the earlier stages of my sickness had been subject to violent fluctuations) showed a heavy, labored action, and there was great difficulty in breathing. I sat up in bed and in a few minutes the symptoms disappeared, and I went to sleep again only to wake up a little later with a repetition of the same trouble. I sat up in a chair for an hour or so, which seemed to relieve the difficulty. When I went to sleep for the third time, I again woke up with increased

symptoms of the same nature, and this time there was no cessation. The lips were dry and the throat sticky, showing a pharyngeal catarrh, the heart was increasing in speed, finally the pulse running up to 116, and a feeling of pressure was manifesting itself over the head. I started to find a physician, reaching the office of Dr. Hardenberg, of Jersey City, about two o'clock. By this time it was all I could do to stagger. The physician came home with me, and was at my bedside for nearly five hours. No other explanation being at hand, the theory of atropine poisoning, from accumulative effect, was adopted and the treatment determined accordingly. Sixty minims of morphine were injected. During the greater part of the next day, I was in an exceedingly nervous condition bordering on delirium. The following day, however, I was entirely over the attack.[1]

[1] How shall we explain this sickness. I do not propose to explain it. I simply propose to present some facts. I began using atropine November 10th, and the predominant feeling in the eyes was one of heaviness. This would occasionally be broken by what I cannot otherwise describe than as a feeling of "give," and a feeling of rest would pass over the eyes, at which time the entire general feeling would change instantly. The transition of feeling was so great, that, as I remarked to Dr. Wilmer in a letter, I involuntarily broke out in a whistle. I cannot explain this feeling of "give" as anything else than the cessation of spasm, and the alteration in general feeling was so instantaneous that it has left a reasonable doubt in my mind whether it went through an alteration of cerebral circulation or was merely the withdrawal of nerve-strain by the stopping of spasm.

Tests taken of the muscles during the last week of November showed

Afterward, under mydriatic, I found the entire relaxation of the posterior meridian to be plus 3.50 D, and the hyperopia plus 1.62 D[s]; (1.62 from 3.50 equals 1.88, coming within an eighth of the reading of the ophthalmometer. Dr. Balmer read my eye in two different instruments, getting a little on each side of 2 D[c]).

FAMILY HISTORY.

The father died of congestion of the brain in March, 1892. Throughout his life he betrayed evidences of nervous irritability. In 1872 he broke down with nervous prostration and was doctored for nearly two years for a supposed threatened attack of softening of the brain. At this time it was impossible for him to read at all. Photophobia appeared in its most severe form and it was necessary to wear the darkest kind of lens. For nearly a year it would produce vertigo to so much as attempt to read the letters on the sign-boards along the street. There was a predominant feeling of approaching insanity. He recovered from this only to break down again in 1880. His eyes again troubled him, the sickness was progressive,

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them balanced, and tests taken December 10th showed them standing as herein given. That is, in that interval I had managed to break the cramp of the ocular muscles and stops spasm. I think it not an illegitimate assumption to suppose that I did so on the night of December 1st, no matter what may or may not have been the concomitant constitutional disturbance.

throughout there was a difficulty of accommodation (see "Cerebral Hyperæmia," William A. Hammond), enervation, insomnia, quivering right eyelid, paralysis of right eyelid, facial paralysis of the right side, partial paralysis of the leg, glaucoma, and finally cerebral congestion, of which he died. There was to be traced through life a sexual weakness, and he was much troubled with light flashes before the eyes.

The mother died of congestion of the brain after having had three attacks. During early life there had been a history of enervation, periodical headaches, and a pain in the side attributed to female troubles. The first attack of congestion antedates my memory. In the early eighties the eyes began to trouble her severely, making it painful to read after dark. Presbyopic glasses were used, which only ameliorated the trouble for a time. Previous to the second attack of congestion complaint was made of a very marked blurring of vision, light flashes before the eyes, quivering of the eyelids and a premonition of approaching insanity after fatigue.

An aunt, sister of the mother, has lately had an attack of cerebral congestion, falling unconscious in the street. She has been troubled through life with a pain in the head and side, and four years ago was complaining of a blurring of vision.

Only a few things more I desire to mention.

While under atropine in November, and previous to

the fuller correction of the cylinder, I was using my eyes constantly with the opthalmoscope, under the old correction of a plus .75 cylinder. The relaxing effect of the atropine, combined with the strain, induced a severe spasmodic oscillation. I desire to simply call attention to one occasion. I retired to bed feeling worse than I had felt for many years, there were light flashes before the eyes, an increased premonition of approaching insanity, a feeling of approaching delirium, a confused feeling in the head and the old gnawing sensation in the neighborhood of Meckel's ganglion. I got up, lit the gas. Objects were blurring badly. I had on hand my old glass plus 2 D^s plus .75 D^c axis 100° in both light and dark. I doubled them, equals plus 4 D^s plus 1.50 D^c, axis 100°, and gazed steadily at my finger. The blurring ceased, the gnawing sensation in the neighborhood of Meckel's ganglion, the delirious feeling, ceased, the confusion stopped, and I went back to bed and went to sleep. The sensation in the eye itself was nothing.

Another thing: When I went into the hands of the first nerve expert March 1, 1887, the pupil of the left eye was smaller than the right, and it always dilated slower and contracted quicker under atropine. Since the relaxation of the latent astigmatism the pupils contract equally.[1]

[1] I have seen reason to believe that the pupil is probably held by the cramp of the ocular muscles.

Its bearing on accommodative myopia and strong tonic spasms is obvious. Given a corneal astigmatism, the posterior meridian cramps to neutralize this, the anterior meridian cramps to obtain a harmonious action of the muscles and strong spasms are the natural consequence. Any attempt to relax the accommodation without correcting the astigmatism is an attempt to produce a non-harmonious action of the muscles.

I noted the operation in my eyes. They were well performed and the eyes balanced almost to a dot. Since the increase of the cylinder tests taken December 10, 1893, under O.S. equal plus 3 D^s minus 2 D^c axis 10° show:

	THIRTY FEET.	TWELVE INCHES.
Esophoria	1°	..
Exophoria	..	10°
Power internal recti muscles	25°	20°
Power external recti muscles	6°	17°
Power inferior rectus right eye	3°	...
Power inferior rectus left eye	4°	5°
Power superior rectus right eye	4°	4°
Power superior rectus left eye	3°	3°
Hyperophoria	3° right eye up.	..
Hyperophoria	.. right eye down	2°

Its implication on the lack of balance of the ocular muscles by reflexing along the third nerve are obvious.

The above views are not set forth as the panacea of all evils. They are simply recitals of what I have seen take place. My own views are that this latent astigmatism

may form an initial centre of irritation upon the branch of the third cerebral nerve, reflexing to almost any part of the nervous system. From demonstration, I can say I have seen this latent astigmatism reflex in

Cerebral hyperæmia,	Lack of balance of ocular muscles,
Melancholia,	Neurasthenia,
Vertigo,	Dyspepsia,
Photophobia,	Stricture of throat,
Sexual weakness,	Palpitation of heart,
Insomnia,	Cephalalgia.

And I have seen reason to believe that it may produce

Cataract,	Delirium,
Facial paralysis,	Mania,
Glaucoma,	General paralysis.

What percentage of such cases it solves is a question for future investigations.

Only one word of caution, which I trust I may be pardoned for giving: that extreme care should be used in administering atropine to a patient with any nervous trouble or a cerebral hyperæmia. I have seen a two-grain solution inside of five minutes throw a patient into hysteria for an hour; have seen it send a shooting pain to the heart and cause the arms and legs to jerk like with chorea.

The justification for the above article is neither a deep knowledge of literature, nor an extensive experience; but that it is original.

I trust that the fact that had it not been for my sickness, I should have already been a member of the medical fraternity, and as it is, expect to be one day, will be allowed to compensate for the fact that I am a layman.

JERSEY CITY, N. J.
January 20, 1894.

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[1] He had used atropine time and again, once when fitted for glasses, four grains to the ounce of water, three times per day for five days.

Under my direction he had been using atropine two grains to the ounce for two weeks prior to the examination, twice per day for the first week and once per day the second week. He appeared for examination December 13, 1893, wearing glasses of the last oculist,

O. D. plus 1 D^s, plus .25 D^c, axis 75°
O. S. plus 1 D^s, plus .50 D^c, axis 105°

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[1] To illustrate the twisting of this spasm, I insert the case of Mr. R. (one of the cases eliminated from the foregoing chapter), who had been in the hands of three oculists, and the tests taken by the usual methods, of lens, ophthalmoscope, and retinoscope.

It shows the method formulated for testing and proving the correctness of ophthalmometric readings, to continue the use of atropine and crowd the eyes with both spherical and cylinder.

The ophthalmometer showed

O. D. .87 D^c, axis 80°
O. S. .87 D^c, axis 105°

His eyes accepted

O. D. plus .50 D^c, axis 85°
O. S. plus .75 D^c, axis 85°

Atropine was continued once per day and the eyes crowded with both spherical and cylindricals. By December 20th, they showed

O. D. plus .75 D^c, axis 80°
O. S. plus .75 D^c, axis 105°

JERSEY CITY, N. J.
January 20, 1894.

II.

THE OCULAR SPASM.

On the 9th day of December, while lighting the gas, a jerk was felt in the internal recti muscles of both eyes. This caused me, on the following day, to take a test of the balance of the muscles, as given in the foregoing article (which test is not to be regarded as final, as ocular errors were latent). The pain in the two muscles continued for several days, and on the following Tuesday, December 12th, it was decided to suppress vision of the right eye. A piece of ground glass was accordingly placed before the eye, and in two hours the pain ceased. The operating oculist was informed of this fact, and also the abnormal balance of the muscles. He immediately replied, requesting me to reopen vision. As the atropine passed out of the eyes a slight amount of blood was noticed in the capillaries of the sclerotic coat. For some reason the eyes did not accept the glass as readily as expected, and atropine was used several times subsequently. Each time as the atropine passed out the blood was noticed, and each time becoming more marked. Atropine

was used for the last time (before the last sickness) January 18th ; and on January 31st, in the afternoon, the capillaries of the sclerotic coat, stretching from the conjunctiva to the cornea on the inner side of the left eye, were noticed to be muddy with blood.[1] No attention was paid to this. That night, while lying in bed, a slight pain was felt over the left eye, which would have passed unnoticed but for the fact that the heart was observed to give a jump at the same time. I hastily arose and lit the gas. The left arm had turned numb, and the right leg was jerking so that it could not be held to the floor. The right arm was shaking. As I gazed into the mirror the eyelids began to quiver and close. The pupils of the eyes were seen to be rapidly dilating and contracting.

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[1] For the purpose of diagnosis, it becomes important to determine whether this bloody appearance was due to inflammation or to mechanical irritation. I think I can safely answer in favor of the latter explanation. I noted that it had appeared before as the atropine was passing out and always in accommodation ; that is, at such a time as there would be a strain upon the converging muscles, and, as I noted to Dr. Wilmer in a letter, it would disappear on looking at a distance, and its disappearance would be accompanied by that same peculiar feeling of "give." Had this been due to inflammation, I cannot conceive that it would have vanished on looking at a distance, or that its disappearance would have been accompanied by a feeling of "give." I regard it simply as due to the mechanical irritation from a spasm of the internal rectus muscle.

[2] It becomes important to determine, if we can, to what to attribute this spasmodic dilatation and contraction of the sphincter muscles. That I think I have satisfactorily done by simply stretching the eye apart with prisms and artificially producing it.

Under the impression that the accommodation had passed into a spasm, atropine was hastily injected, a solution of two grains to the ounce being used. This was used for three days and nights, six times a day, with the symptoms becoming constantly worse. There would be heavy jerks upon the internal recti muscles of both eyes. The light flashes before the eyes formed a perfect aurora borealis, in which all manner of red and yellow lights wound themselves in fantastic confusion. By way of parenthetic remark, I here suggest that this may offer possible explanation of why a man in delirium tremens thinks he sees serpents.

The nervous and mental symptoms got continually worse. On Saturday night, with no glass before the eye, the right hand was accidentally placed over the right eye. Immediately the symptoms improved. Vision was then suppressed in the right eye, and the correction used on the left eye; but by Sunday noon, as the improvement did not seem sufficient, vision was again opened in the right eye, the theory being entertained that the irregular astigmatism of the right eye had caused the trouble. All the symptoms immediately became worse — the light flashes, the iron band around the head, accompanied by pressure and pounding upon top, and the jerking of the various muscles, causing a shaking and vibration to all objects viewed.

Monday morning, as I gazed into the mirror, I felt a

heavy tug at the two internal recti, accompanied by a sledge-hammer blow upon the top of the head. The predominant feeling was to fall over upon the floor and yell. As it was, I fell over in a chair with a heavy congestive chill, which lasted for about twenty minutes. While right in the midst of this chill, the atropine was again injected, and, with the naked eye fixed upon the test-card, a blind was placed over the right eye. Not in ten minutes, nor in five minutes, but *instantly* the accommodation of the left eye was seen to relax, and the chill ceased.

The theory then formulated, and since found to be correct, was, that the abnormal convergence had dragged ahead the accommodation of the left eye, making it impossible at that time to accept the glass. Vision was again suppressed in the right eye, mydriatics almost wholly discontinued, and the left eye gradually worked up to the glass, as the divergence of the muscles permitted.

Other facts point to the view that the abnormal convergence had initiated the spasm of the accommodation.

The last three years my left eye has been unable to accept any spherical, although wearing glasses constantly. A consideration of the manner in which the operations were performed suggests the immediate explanation. Although the oculist was very careful to fit the eyes before operating, being thoroughly aware of the accommodation affecting convergence, after the glass was fitted, the atro-

pine was allowed to go out of the eye, and the ocular and ciliary muscles to recramp. The consequence of this was that the glass before the eye, during the operation, had no other effect than the prismatic power of its lens; and the penalty, at any time in the past three years, for accepting the glass, has been to become exophoric.[1]

The views which we reached *a priori*, from the nature of the operation, are sustained *a posteriori* from the condition of the muscles. Having vision suppressed in the right eye for two weeks, the eyes show a heavy exophoria and a heavy hyperophoria with the very glass under which I was operated on.

And another fact points to like conclusions. Upon the theory that the ciliary muscles were weak, some nine or ten months ago, one dioptric spherical was added to each lens; but although wearing this for nearly a year for reading (which, to the normal eye, would amount to no more than a weak presbyopic glass), the eyes refused to accept them comfortably. Last October, while reading

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[1] The above criticism is theoretically correct and there is no doubt of the fact that the abnormal convergence created by the operations served to solidify the ocular cramps and to hold the accommodation; still, as a matter of justice to the physician, knowing what I do of the tenaciousness of these cramps, I am not prepared to assert that they would necessarily have passed out had the operations not been performed. All that can be asserted with emphasis is, that were it possible for them to pass out, the abnormal convergence created by the operations destroyed the opportunity.

with a nine-degree prism in the hand, I happened to place it, base in, over one of the eyes. Instantly the eye accepted the glass with comfort.[1]

After the suppression of vision in one eye, the fluttering of objects assumed a tolerable regularity. They were jerked to the right in the left eye, to the left in the right eye, and jerked down in the left eye, showing that certain muscles of the eyes were endeavoring to act in conformity with the relaxed accommodation, but that a spasm of other muscles prevented them from doing so. At a distance of about four feet a picture-frame, some $4\frac{1}{2}$ inches wide, was seen solidly as about 9 inches wide. This gives some idea as to the amplitude and velocity of the vibrations.

When we remember that a visual image remains on the retina about $\frac{1}{16}$ of a second, and if reproduced every $\frac{1}{16}$ of a second it shows a constant image, we are forced to

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[1] Further consideration has convinced me that the above is a faulty explanation. The glass I was wearing was O.S. plus 3, D^s plus .75, D^c, axis 100°, O. D. plus 5 D^s, and the eyes stood manifest O.S. plus .75 D^c, O. D. plus 2.25 D^s. I had really simply procasted vision, that is I had thrown my accommodation and convergence in such a position as if viewing a distant object while the lens and prisms enabled me to view a near one.

This leads to the consideration of another error, the injudicious use of heavy unaccepted sphericals, constituting

1st. A blur on the retina.

2d. A continued pressure on the ocular muscles.

3d. Entangling accommodation with convergence.

conclude that the vibrations of the eye must have been in the neighborhood of one thousand to the minute, with an amplitude of four or five degrees.[1]

After the oscillations had slowed down somewhat, objects appeared to move with the rapidity of the shuttle of a fast-running sewing-machine. These oscillations have now been almost stopped, and under no other treatment than suppression of vision in the right eye. The cause being removed, the effect is ceasing.

I should have remarked before that, for several days, there was a partial paralysis of the throat and tongue, making it difficult to speak and swallow, without effort.

With vision suppressed in the right eye, when looking through a heavy spherical glass on the left eye, there would be heavy jerks on the external recti muscles of both eyes, showing an effort to diverge in conformity with the relaxed accommodation of the left eye; and at such times a numbness would be felt in the forehead, sometimes running down the sides of the face into the jaw.

In the previous article reference was made to the fact that, when I visited Dr. Richey, objects were seen shaking and vibrating. I was led there to attribute this wholly to

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[1] The validity of the above calculation depends on the length of time a visual image remains on my own retina. It varies in different individuals, and I have never tested my own. I have simply assumed the average.

the spasms of accommodation. I now am led to believe that seven years ago I also had an ocular spasm. What percentage of the blurring is to be accounted for by the spasm of accommodation, and what percentage by a spasm of the ocular muscles, I am unable to say.

Now, some six weeks ago, while under the heavy use of atropine, I was able to demonstrate that a manifest, compound, hyperopic astigmatism may produce diplopia in one eye. In the case of my father it was the same. Some years before his death, he called me to his desk, and, with his hand over his left eye, said that the type would seem to run ahead of one another, when viewed by his right eye. We must conclude, then, that in this case a latent astigmatism was relaxing. In conformity with that relaxation the eye desired to turn out, but an abnormal strain of convergence held it in; and to this fact we may attribute the paralysis of the internal rectus muscle. And to the fact that the accommodation wished to relax, while he held it contracted by his efforts to read, we may probably assign the cause of his glaucoma.

The above sickness has demonstrated several things:

1st. That the drawn sensation, often complained of by persons in their eyes, may be due to a tension of the ocular muscles under spasm.

2d. That the light flashes in the eyes may be due to the mechanical pressure on the eyeballs, from the jerking of the ocular muscles.

The above views are offered, not with the idea that they contain a solution of all the phenomena at hand, but with the view that they contain an adumbration of truth, pointing to the opening up, rather than to the closing, of a field of investigation. What percentage of nerve-strain is due to the spasm of the ciliary muscles, and what percentage is due to a spasm of the ocular muscle, it is impossible for me to say; nor am I placed in a position where I can investigate that matter. It is, therefore, accordingly offered to the consideration of those who are in a position to give it their attention.

JERSEY CITY, N. J.
February 26, 1894.

APPENDIX.

From May 1, 1894, to the middle of November of the same year, I underwent treatment, which, whatever may have been its constitutional effect, had a local effect upon the ocular muscles and caused a fluctuation in both the exophoria and hyperophoria. No accurate tests were taken but the exophoria probably reached ten degrees. At the present time, with treatment suspended, the muscles have assumed for distant vision—

Esophoria less than 1°
Hyperophoria less than $\frac{1}{2}$°

and the accommodation has cramped to

O. S. + 1.50 D^c, axis 95°
O. D. + 2.25 D^s

The irregular astigmatism of right eye disregarded, which gives complete relief.

But the effect of the treatment has been to leave the ocular cramps elastic. I have performed a series of experiments which I think both interesting and instructive.

Experiment I.

Copied from my notes of January 22, 1895.
The tug being absent.

Δ s 2° base in, over the eyes for five minutes and I get the following sensations :

1. Tug at internal recti muscles.
2. Slight chills.
3. Face flushes.
4. Very slight sinking sensation at stomach.
5. Numbness, left jaw.
6. Severe twitching facial muscles and quivering eyelids.
7. Gnawing sensation left side, neighborhood Meckel's ganglion.
8. Sharp shooting pains through base of brain, almost cause fainting.
9. Respiration and heart slightly affected.

How shall we explain this? As stated above, the internal recti have contracted. But the backbone of the cramp has been broken. It is no longer rigid but elastic. The effect of the abductive prisms is to turn the eyes out, increasing the tension of the ocular muscles. Gradually the elastic cramp yields, goes into a spasm, producing the tug and the other symptoms are reflexes from that spasm.

Experiment II.

Copied from my notes of March 5, 1894.

Atropine had been out of the eyes about a month, the ocular muscles had cramped somewhat, though they were still in a very elastic condition. I had been wearing O. S. + 2 D^c, axis 95; O. D. ground glass. I placed this formulæ before the eyes: O. S. + 1.50 D^s + 2 D^c, axis 95; O. D. ground glass, and gazed around the room. In a few minutes it caused a heavy jerking on the external recti muscles which caused not only the arm but the flesh on the chest and leg to turn numb and caused me to limp.

How shall we explain this. It seems to me the only explanation is that the spherical pressed back the latent accommodation, caused the eyes to attempt to diverge and pulling against an elastic cramp of the ocular muscles, reflexed down.

Experiment III.

Copied from my notes, February 26, 1895.

When I went to bed, twitching over the body, slight swaying head, tugging at internal rectus muscle and light flashes very severe, insomnia for about an hour, suddenly there is a heavy jerk upon the internal rectus muscle, the

head gives a heavy sway, a throe goes over the entire body, *simultaneously* the tug and the light flash stop and in a few minutes I fall asleep.

How shall we explain this? It seems to me that the only logical explanation is that during the day the muscle has been stretched out. When I go to bed it begins to take up its relaxation, sways back and forth in spasm, producing the tug, the light flash and the general spasmodic symptoms, when I get the heavy jerk, the muscle has cramped up its maximum of spasm, the conflict is over, equilibrium has been restored and nature sinks to rest.

Experiment IV.

From notes, March 15, 1894.

It was on March 15, 1894, that tests were taken of my eyes by a well-known New York oculist. The method pursued was to use a bar of prisms, in ascending series, and rapidly run them before the eyes. When they came up to the high numbers, they would come up with a jerk and the reflex was so severe as to cause the arms and legs to jerk, raising the feet from the floor. Furthermore, the reaction was such for several days as to cause face flushes and a deep sighing.

Experiment V.

Copied from my notes of March 19, 1895.

After a hearty meal, I experienced the following sensations: tugging internal recti muscles, numb left jaw, slight numbness left arm, and chills almost convulsive. There was absolutely no nausea and no sensation at the stomach. Now by referring to Experiment I., it will be seen that I have been able to produce chills, numb left jaw, and tug of the muscles by prisms; and by referring to Experiment II. it will be seen that I have been able to produce a numb arm by the simple pressure of a heavy unaccepted spherical. That is, I have been able by purely mechanical work upon the eye to produce all the symptoms I experienced on this occasion, and the legitimate inference is that I experienced the symptoms from the same cause, only reaching them by different means.

How shall we explain it? As noted above, the backbone of the cramp is broken. It is no longer rigid but elastic. It is in a condition of unstable equilibrium, still contracted but ready to relax on the slightest provocation. The draft upon the nerve energy from the hearty meal sends it into spasm, and the other symptoms are reflexes from that spasm.